EXHIBIT

21

DEFENDANTS' MOTION TO EXCLUDE THE TESTIMONY OF DR. CHRISTOPHER TEAF

05-CV-0329 GKF-PJC

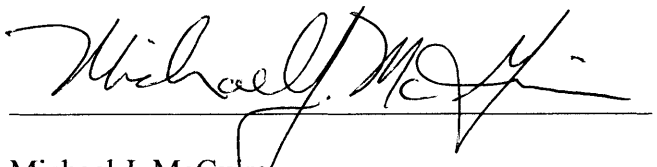
United States District Court
Northern District of Oklahoma

Expert Report of Michael J. McGuire, PhD, PE, BCEE

January 26, 2009

Prepared for

State of Oklahoma, et al. v. Tyson Foods, Inc., et al.
Case No. 4:05-cv-00329-GKF-PJC



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> carcinogenic substances that may occur in treated drinking water supplies (Cooke and Welch, 2008)."

As demonstrated in the previous section, Cooke and Welch did not demonstrate any association between the spreading of poultry litter and levels of DBPs in tap water.

Sources of DBPs and Organic Matter

On page 23, Teaf (2008a) made the same mistake made by Cooke and Welch (2008a) by attributing all of the organic materials that contributed to the formation of DBPs to runoff from fields where poultry litter had been applied and to the increased productivity of the river and reservoir caused by algae blooms stimulated by higher phosphorus levels. He ignored important sources of organic carbon as noted on Figure 3 in my report that includes natural organic matter from leaves, soil and other naturally occurring organics and the presence of DBP precursors in the organic fraction discharged by wastewater treatment plants.

On page 24, Teaf (2008a) stated, "The formation of DBPs is correlated significantly with the content of dissolved organics in raw water…" Teaf dramatically oversimplifies the role played by TOC in production of DBPs. As already stated in my report, the production of TTHM and HAA5 is based on far more than just the organic material in the water. Figure 18 shows that raw water TOC data from the 296 ICR utilities is NOT correlated with TTHM concentrations in the treatment plant finished water across the U.S. (McGuire and Graziano 2002). The reason for this lack of correlation is important and easy to explain. Those utilities in the U.S. that had high levels of TOC in their sources of supply made treatment changes that mitigated the production of THMs in their distribution systems so that they could comply with the DBP regulation. Also, many of the treatment and distribution systems used by the 500 ICR plants were quite different and produced different DBP levels even if the raw water TOC levels were similar. For example, utilities with relatively low TOC produced high levels of TTHM most likely due to high doses of chlorine and long contact times.

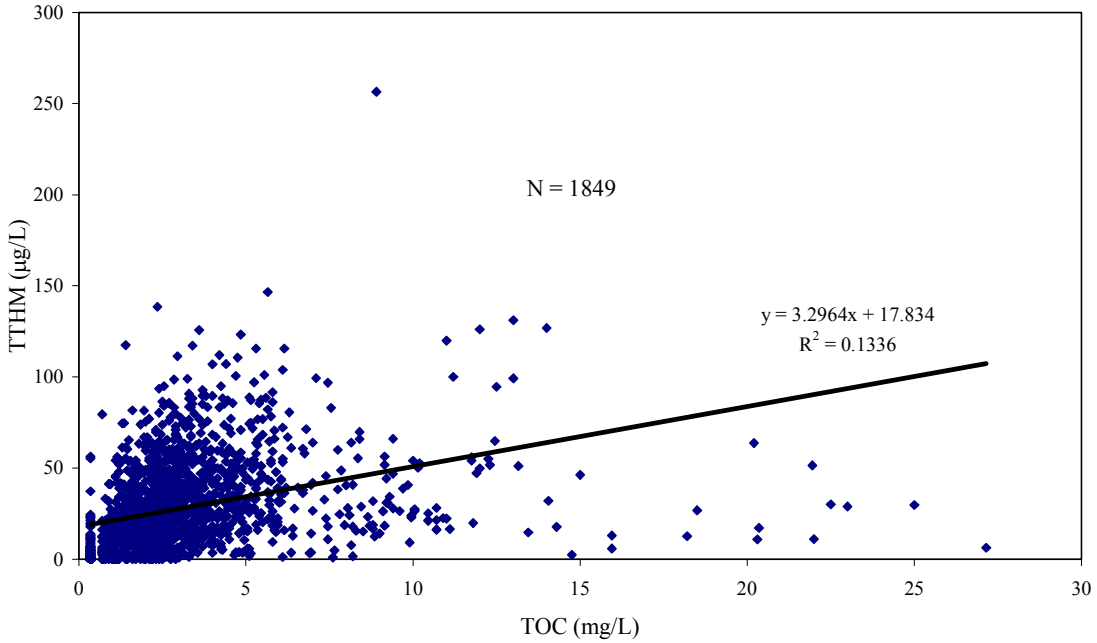

Figure 18. Raw Water TOC Concentrations Versus Finished Water TTHM Concentrations for ICR treatment Plants (McGuire and Graziano 2002)

Later on page 24 of his report, Teaf (2008a) attempts to connect the spreading of poultry litter with high levels of DBPs. As did Cook and Welch, Teaf fails to make the connection using real data from the IRW watershed and utilities treating water from the Illinois River and Lake Tenkiller. Instead he attempts to make the connection citing other studies, which proposed that elevated nutrient inputs were related to DBP production in other watersheds and treatment plants. He strings together a list of suppositions and unsubstantiated statements to try to prove his argument. He failed to prove any connection between the spreading of poultry litter in the IRW and levels of DBPs in water utility distribution systems in the IRW.

Errors Comparing DBP Data with MCLs, MCLGs and Chloroform Risk Based Screening Level

On page 26, Teaf (2008a) gives a synopsis of the Stage 1 and 2 DBP rules. He correctly lists the MCLs and MCLGs for DBPs regulated under the Stage 2 DBPR. However, he then lists "…the following restrictive water concentrations were identified by USEPA (2006c) as being necessary to meet the standard regulatory benchmark of 1-in-one million cancer risk…" Teaf's statement is incorrect. There is no mention of utilities having to meet a one-in-one-million cancer risk in any part of the Stage 1 or Stage 2 DBPRs or supporting documentation. Also, water utilities are not required to comply with the concentrations listed for TTHM and HAA5 MCLGs. MCLGs are goals only.

In Teaf's deposition (Teaf 2008c, page 393), he again identifies the MCLG as a regulatory limit:

> **"Q** I want to go through the columns [Table T1] and clear up a few things for me. I know you've testified about this extensively yesterday and I'm trying not to plow the

same ground. Under chloroform, there are two columns. There's the MCLG of 70 micrograms per liter; right?
A Yes.
Q Excuse me. Is that a regulatory limit?
A For chloroform?
Q Yes, sir.
A Yes.
Q It's a regulatory limit that cannot be exceeded in treated water?
A The MCLG and the MCL in the case of chloroform are equivalent to one another."

As mentioned many times in my expert report, water utilities are required to meet MCLs based on the monitoring requirements and calculation methodologies incorporated in the regulation. Meeting MCLs is the foundation for compliance with primary drinking water regulations under the Safe Drinking Water Act. The MCLG and the MCL in the case of chloroform are absolutely NOT equivalent to one another. Teaf's assertion that utilities must comply with numbers, goals and levels other than MCLs is flatly wrong.

Teaf (2008a) carries his mistake forward when he introduces the "risk-based screening level for chloroform" at 0.17 ppb. He misuses the screening level for chloroform which was developed by the USEPA along with the other (more than 100) screening levels to determine relative risks associated from exposure to environmental contaminants at hazardous waste disposal sites (USEPA 2007a). In other words, the 0.17 ppb risk-based screening level would normally be compared to chloroform levels in residential water that is contaminated with chloroform that resulted from the illegal or uncontrolled disposal of hazardous wastes containing chloroform (along with other chemicals). The 0.17 ppb screening level was never recommended to be compared to chloroform values associated with drinking water chlorination. As stated previously in my report, regulation of THMs (including chloroform) is based on a balancing of risk that includes the benefits associated with elimination of microbial disease by chlorination of drinking water (Murphy and Craun 1999).

On page 34 of the Background document for the screening levels (USEPA 2007a), the USEPA authors stated problems that have been noted in the misuse of the risk-based screening levels:

"Potential Problems:
As with any risk-based tool, the potential exists for misapplication. In most cases the root cause will be a lack of understanding of the intended use of the screening levels table. In order to prevent misuse of screening levels, the following should be avoided:

- Applying screening levels to a site without adequately developing a conceptual site model that identifies relevant exposure pathways and exposure scenarios.
- Not considering background concentrations when choosing screening levels.
- Use of screening levels as cleanup levels without the consideration of other relevant criteria…" (USEPA 2007a)

The intention of this quoted list of problems is clear, but Teaf (2008a) pays no attention to the limitations embodied in the "Potential Problems" list.

Further, the screening level values were never anticipated to be regulatory limits under any circumstances. Clearly stated on page 2 of the background document is a caution against the improper use of the tabulated values. "The table was not generated to represent action levels or cleanup levels but rather as a technical tool." Teaf ignores this caution and misuses the chloroform 0.17 ppb screening level.

Teaf compounds his lack of understanding of DBP regulatory history and practice when he stated at the bottom of page 27 (Teaf 2008a), "Stated simply, if risks of this magnitude were found at a waste disposal site or an industrial contamination site, in my experience they would require attention and remediation." He betrays his ignorance of drinking water treatment and regulatory compliance by comparing waste disposal site risk analyses with drinking water regulatory practices by the USEPA and state primacy agencies.

The incorrect and non-scientific basis that Teaf used to compare the chloroform risk-based screening level of 0.17 ppb with chloroform concentrations in drinking water served by IRW-based water utilities was improper and should not be considered.

Error Comparing THMFP and TTHM Data

On page 28, Teaf (2008a) discussed THMFP results collected by CDM. Teaf's Table T3 (Teaf 2008a) summarizes the THMFP results from five of the twelve locations sampled by CDM. Teaf states, "…71% of the results (57/80) showed values [of THMFP] in excess of the TTHM MCL at twelve locations along the Illinois River and in Lake Tenkiller." A footnote to Table T3 refers to average THMFP data in the body of the table and states, "*Exceeds EPA drinking water standard of 80 μg/L for Total Trihalomethanes."

Teaf (2008c) stated in his deposition on page 380 in answer to a question:

> **"Q** All right. Now, tell me what does trihalomethane-forming potential tell you about the raw water source sample.
> **A** It tells you the inherent ability of that water to form trihalomethanes upon a **normal** chlorination process." (emphasis added)

It is completely improper for Teaf to compare THMFP values with the TTHM MCL. Such a basic mistake demonstrates a lack of understanding of what THMFP means and how it is used by water quality professionals. The THMFP value is determined by a specific analytical method (APHA 2008). A large dose of chlorine is added to a sample of water so that a chlorine residual of more than 3 mg/L can be detected after seven days at a temperature of 25 degrees C and a pH of 7.0. The chlorine dose could be 6 to 10 mg/L to achieve a greater than 3 mg/L chlorine residual after seven days. These testing conditions do NOT represent a "normal chlorination process." They are designed to accelerate the production of THMs to give an indication of organic and inorganic (bromide) precursor levels in the raw water.

Cooke and Welch (2008a, page 16) also mistakenly compared THMFP with the TTHM standard stating, "All nine utilities had a THMFP in excess of the THM violation standard of 80 µg/L set by the USEPA."

THMFP is reported as chloroform equivalents. As summarized from APHA (2008), the formula for calculating THMFP as chloroform equivalents is:

$$THMFP = A + 0.728B + 0.574C + 0.472D$$

where:
A = µg /L chloroform
B = µg /L bromodichloromethane
C = µg /L dibromochloromethane
D = µg/L bromoform

As noted, the concentrations of each of the three THMs other than chloroform are converted to an equivalent chloroform concentration by multiplying the analytical concentrations of each by a ratio of the molecular weight of chloroform to the molecular weight of that compound.

TTHM which is used to determine compliance with the MCL is calculated by summing the µg/L concentrations of the four THM components without any conversion to equivalent chloroform concentrations. Therefore, the basis for calculating TTHM and THMFP are completely different and cannot be compared. However, Teaf (2008a) made an even more fundamental mistake when he compared THMFP values with TTHM data collected from a distribution system as part of TTHM regulatory compliance.

On page 35 of a book devoted to DBPs, Xie (2004) stated categorically:

> "Formation potential test [THMFP] is a procedure to evaluate the DBP precursors rather than the formation of DBPs in finished water…However, the **DBP formation potential results cannot be used to estimate the DBP formation under actual chlorination conditions**." (emphasis added)

THMFP is a measure of the potential for a water sample to form trihalomethanes under extreme chlorination conditions in a laboratory environment. Comparing THMFP values of raw water with the TTHM MCL or levels in a utility distribution system is completely improper and an egregious error. Any conclusions or expert opinions from Teaf that are based on his comparison of THMFP data and the TTHM MCL should be ignored.

Error with OWRB Surface Water Criteria

On page 28 of his report, Teaf (2008a) states:

> "Also found in that criterion is OAC 28 785:45-5-10(5) (B) which states: 'These waters shall be maintained so that they will not be toxic, carcinogenic, mutagenic, or teratogenic to humans' (OAC, 2007). The reported TTHM and HAA5s concentrations detected in IRW waters clearly demonstrate that this criterion is not being met."